

Cardiac Curvature on the Beat-Pair Torus:

Geometric Diagnosis of Heart Failure, Arrhythmia, and
Atrial Fibrillation from Pulse Timing Alone

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Paper I in the Cardiac Torus series

March 2026

Abstract

We introduce the Cardiac Ramachandran Diagram, a geometric framework that maps consecutive heartbeat intervals onto a torus (T^2) and computes geodesic curvature of the resulting trajectory. By analogy to the Ramachandran plot in protein structural biology—where consecutive backbone dihedral angles reveal fold geometry—consecutive RR intervals define angular coordinates on a flat torus whose curvature encodes the dynamical state of the cardiac rhythm.

Across 107,531 annotated beats from the MIT-BIH Arrhythmia Database, torus curvature separates the four major arrhythmia classes (Kruskal-Wallis $H = 9,917$, $p < 10^{-300}$). Ventricular ectopic beats exhibit median curvature 6.6-fold lower than normal beats ($\kappa = 0.99$ vs. 6.55 , rank-biserial $r = -0.70$), reflecting ballistic excursions that bypass autonomic feedback. Extending to 184 records across five PhysioNet databases, two torus-derived parameters—median curvature (κ) and curvature Gini coefficient (G_κ)—create a diagnostic feature space separating congestive heart failure ($\kappa = 32.0$), normal sinus rhythm ($\kappa = 10.0$), atrial fibrillation ($\kappa = 3.3$), and ventricular arrhythmia ($\kappa = 1.2$) with pairwise effect sizes $|r| > 0.71$.

CHF replication across 116 records from four independent cohorts confirms a dose-response gradient: NYHA class 1–3 patients show intermediate curvature ($\kappa = 20.1$), positioned between normal ($\kappa = 10.0$ – 16.4) and severe NYHA 3–4 ($\kappa = 32.0$), with curvature tracking disease severity ($r = 0.61$, $p = 0.001$). Noise robustness testing demonstrates that record-level diagnostic separation survives photoplethysmography (PPG)-realistic timing jitter: at $\sigma = 20$ ms, the Normal-Ventricular Arrhythmia effect size remains $|r| = 0.95$ ($p < 10^{-50}$).

The framework requires no ECG morphology, no frequency-domain analysis, and no machine learning. It operates on pulse timing alone, making it deployable on any device that measures the interval between heartbeats—including consumer PPG wearables. Two numbers from a donut may upgrade every fitness tracker on the planet to a cardiac screening device.

1. Introduction

1.1 The Wearable Cardiac Monitoring Gap

Consumer wearable devices have transformed cardiac health monitoring. The Apple Watch, Samsung Galaxy Watch, Fitbit, and similar devices can detect atrial fibrillation (AF) using photoplethysmographic (PPG) sensors and, in some cases, single-lead ECG electrodes. These capabilities have produced FDA-cleared screening tools that have identified previously undiagnosed AF in hundreds of thousands of users.

However, a critical gap remains. Congestive heart failure (CHF)—which affects 6.7 million Americans, is the leading cause of hospitalization in adults over 65, and kills more people annually than most arrhythmias—is undetectable by any current consumer wearable. CHF does not produce a distinctive ECG waveform or a pathological heart rhythm; instead, it manifests as a gradual loss of heart rate variability that is invisible to the morphology-based and rhythm-detection algorithms deployed in commercial devices.

Ventricular arrhythmias, supraventricular arrhythmias, and the discrimination between AF subtypes similarly benefit from approaches that go beyond waveform shape. The common thread: the information is in the timing between beats, not in the shape of each beat.

1.2 The Torus Insight

We observe that consecutive inter-beat intervals (RR intervals) naturally inhabit a torus. Each RR interval is bounded between physiological limits (~ 200 – 2000 ms, corresponding to ~ 30 – 300 bpm) and varies quasi-periodically with respiration, autonomic tone, and cardiac pathology. A pair of consecutive intervals (RR_{pre} , RR_{post}) defines a point on the product space $[RR_{min}, RR_{max}] \times [RR_{min}, RR_{max}]$. When each coordinate is normalized to $[0, 2\pi)$, this product space is topologically a flat torus T^2 .

This is directly analogous to the Ramachandran diagram in protein structural biology, where consecutive backbone dihedral angles (ϕ , ψ) define points on a torus that reveal secondary structure and fold geometry. In that domain, the geodesic curvature of the trajectory on T^2 encodes mechanical properties of the protein backbone: high curvature at helix-coil transitions, low curvature within regular secondary structure elements. We hypothesized that the same geometric quantity—geodesic curvature of the cardiac trajectory on T^2 —would encode the dynamical state of the heart.

1.3 Prior Work

Heart rate variability (HRV) analysis operates on the same raw data (RR intervals) but extracts frequency-domain features (LF/HF ratio, spectral entropy) or statistical summaries (SDNN, RMSSD, pNN50). Phase-space reconstruction using Takens delay

embedding into Euclidean R^2 has been explored for arrhythmia classification, with attractor geometry distinguishing some classes at approximately 83% accuracy. The Poincaré plot (RR_n vs. RR_{n+1}) is widely used in HRV research. All of these approaches embed the cardiac signal in flat space, discarding the topological fact that RR intervals are bounded and quasi-periodic. Our framework adds three elements: (1) the correct topology (periodicity in both coordinates), (2) an intrinsic geometric invariant (geodesic curvature), and (3) a concentration measure (Gini coefficient) that distinguishes structured from uniform dynamics.

1.4 Contributions

This paper makes four contributions. **First**, we define the Cardiac Ramachandran Diagram and the mathematical framework for geodesic curvature on the beat-pair torus (Section 2). **Second**, we demonstrate that torus curvature separates four arrhythmia classes with large effect sizes across 107,531 annotated beats, and that quadrant occupancy on the torus reveals the compensatory pause mechanism of ventricular ectopy (Section 3.1–3.2). **Third**, we extend to seven PhysioNet databases encompassing CHF, AF, SVA, VA, and normal sinus rhythm, showing that two parameters (κ , G_κ) separate four cardiac conditions; we replicate the CHF finding across four independent cohorts with a dose-response gradient tracking NYHA class (Section 3.3–3.4). **Fourth**, we characterize noise robustness, demonstrating that record-level diagnosis survives PPG-realistic timing jitter, establishing a two-tier deployment model: PPG for screening, ECG for beat-level classification (Section 3.5).

2. Methods

2.1 Beat-Pair Torus Mapping

For a sequence of annotated beats with inter-beat intervals $\{RR_1, RR_2, \dots, RR_n\}$, we define beat-pair angular coordinates for each interior beat i :

$$\theta_1(i) = 2\pi \times (RR_{\text{pre}}(i) - RR_{\text{min}}) / (RR_{\text{max}} - RR_{\text{min}})$$

$$\theta_2(i) = 2\pi \times (RR_{\text{post}}(i) - RR_{\text{min}}) / (RR_{\text{max}} - RR_{\text{min}})$$

where $RR_{\text{pre}}(i)$ is the interval preceding beat i , $RR_{\text{post}}(i)$ is the interval following beat i , $RR_{\text{min}} = 200$ ms (~ 300 bpm), and $RR_{\text{max}} = 2000$ ms (~ 30 bpm). Intervals outside this range are excluded as physiologically implausible. Both coordinates are clipped to $[0, 2\pi)$, yielding a point on the flat torus $T^2 = [0, 2\pi) \times [0, 2\pi)$. We simultaneously compute three torus representations: Torus A maps (RR_{pre} , RR_{post}), Torus B maps (RR_{pre} , R-peak amplitude ratio), and Torus C maps (RR ratio, amplitude ratio). Results focus on Torus A unless specified.

2.2 Geodesic Curvature on T^2

For three consecutive points $p_{\{i-1\}}$, p_i , $p_{\{i+1\}}$ on the flat torus, we compute Menger curvature:

$$\kappa_i = 4A_i / (|a_i| \cdot |b_i| \cdot |c_i|)$$

where A_i is the area of the triangle (Heron's formula) and a_i , b_i , c_i are side lengths computed as geodesic distances on T^2 with periodic boundary conditions:

$$d(\theta_a, \theta_b) = \sqrt{[\min(|\Delta\theta_1|, 2\pi - |\Delta\theta_1|)]^2 + \min(|\Delta\theta_2|, 2\pi - |\Delta\theta_2|)^2}$$

High curvature indicates the trajectory is turning sharply on the torus, corresponding to active beat-to-beat regulation by autonomic feedback. Low curvature indicates straight-line motion, corresponding to ballistic excursion (ectopic beat followed by compensatory pause) or rigid rhythm (loss of variability).

2.3 Curvature Summary Statistics

For each record or clinical condition, we compute: (a) median geodesic curvature κ ; (b) curvature Gini coefficient G_κ , measuring concentration ($G = 0$: uniform curvature everywhere, $G \rightarrow 1$: curvature concentrated at few beats); (c) curvature bursts—contiguous segments exceeding the within-record 90th percentile, with minimum length 2 beats and merge gap ≤ 2 beats; (d) quadrant occupancy—the fraction of beat pairs in each of four torus quadrants: Q1 (fast→fast, both $RR < \text{median}$), Q2 (fast→slow, $RR_{\text{pre}} < \text{median}$, $RR_{\text{post}} > \text{median}$), Q3 (slow→slow), Q4 (slow→fast); (e) torus spread (standard deviation of angular positions, measuring how much of T^2 the trajectory explores) and torus speed CV (coefficient of variation of beat-to-beat angular velocity).

2.4 Datasets

Seven PhysioNet databases provide the evidence base:

Database	n	Condition	Duration	Analysis Role
MIT-BIH Arrhythmia (mitdb)	48	Mixed	30 min	Beat-level arrhythmia classification (107,531 beats)
MIT-BIH Normal Sinus (nsrdb)	18	Normal	24 h	Multi-disease analysis; CHF replication reference (NSR1)
MIT-BIH Atrial Fib (afdb)	25	AF	10 h	Multi-disease analysis
BIDMC Congest. HF (chfdb)	15	CHF 3-4	20 h	Multi-disease analysis; CHF replication (severe)
MIT-BIH Supra-V (svdb)	78	SVA	30 min	Multi-disease analysis
CHF RR Interval (chf2db)	29	CHF 1-3	24 h	CHF replication (mild-moderate)
Normal Sinus RR (nsr2db)	54	Normal	24 h	CHF replication reference (NSR2)

Total: 267 records. All databases are freely available from PhysioNet (physionet.org) under open-access licenses. The MIT-BIH Arrhythmia Database provides beat-level annotations by two or more cardiologists (~110,000 annotations). The remaining databases provide beat-time annotations from which RR intervals are derived.

2.5 Noise Robustness Protocol

To assess viability for PPG-based wearable deployment, we simulated timing jitter by adding independent Gaussian noise to each RR interval at ten levels ($\sigma = 0, 2, 5, 10, 15, 20, 30, 50, 75, 100$ ms). ECG R-peak localization is precise to ~1 ms; PPG peak detection has reported jitter of 10–30 ms depending on sensor quality, skin tone, and motion artifact. At each noise level, torus curvature was recomputed 10 times to estimate variance. Diagnostic separation (rank-biserial r) was measured at both the beat level (individual AAMI class pairs across all beats) and the record level (Normal vs. Ventricular Arrhythmia classification across MIT-BIH records).

2.6 Statistical Analysis

Group comparisons: Kruskal-Wallis H test (overall) and pairwise Mann-Whitney U tests with rank-biserial effect sizes ($r = 1 - 2U/n_1n_2$). Association: Pearson's χ^2 for quadrant-class contingency tables. All p-values are reported as exploratory without multiple-comparison correction. Effect sizes are the primary measure of diagnostic separation: $|r| > 0.5$ = large, 0.3–0.5 = medium, 0.1–0.3 = small. CHF replication success criterion: $|r| > 0.5$ against the matched normal reference.

3. Results

3.1 The Cardiac Ramachandran Diagram

From 48 MIT-BIH Arrhythmia Database records, we extracted 107,531 beat pairs with valid RR intervals (200–2000 ms). The AAMI class distribution was: Normal 84.0% (n = 90,277), Ventricular 6.6% (n = 7,098), Unknown/Paced 6.2% (n = 6,658), Supraventricular 2.5% (n = 2,697), and Fusion 0.7% (n = 801).

Figure 1 (the Cardiac Ramachandran Diagram) plots all 107,531 beat pairs on Torus A. Normal beats form a tight diagonal ridge in Q1 (fast→fast), reflecting the homeostatic pairing where each interval approximates its predecessor. Ventricular beats explode off this ridge into Q2 (fast→slow), driven by the compensatory pause that follows premature ventricular contractions. Supraventricular beats occupy an intermediate displacement. The beat density on T^2 (Figure 1B) reveals the normal attractor as a concentrated hot ridge, with arrhythmic beats as diffuse halos. The curvature heatmap (Figure 1C) shows that the highest geodesic curvature resides along the normal ridge—constant turning, constant regulatory feedback—while arrhythmic excursions are geometrically flat.

3.2 Curvature Separates Arrhythmia Classes

Overall: Kruskal-Wallis $H = 9,917.5$ (p underflows 64-bit float arithmetic; the four classes occupy distinct curvature regimes). All six pairwise Mann-Whitney comparisons are significant at $p < 10^{-3}$.

AAMI Class	n (beats)	Median κ	Gini	Q2%	Geometric Interpretation
Normal (N)	89,857	6.55	0.532	1.4%	Tight orbit on T^2 ; active autonomic feedback (RSA)
Fusion (F)	799	4.95	0.571	0.4%	Mixed sinus + ventricular; partial trajectory control
Supraventricular (S)	2,675	1.61	0.692	13.0%	Moderate disruption; displaced but not ballistic
Ventricular (V)	7,090	0.99	0.488	23.1%	Ballistic excursion; bypasses autonomic loop

Key pairwise effect sizes: N vs. V: $r = -0.696$ ($p = 0.0$)—a large effect indicating 85% of ventricular beats have lower curvature than the median normal beat. N vs. S: $r = -0.289$ ($p = 10^{-143}$). S vs. V: $r = -0.246$ ($p = 10^{-8}$). V vs. F: $r = +0.670$ ($p = 10^{-212}$).

Interpretation: Ventricular ectopic beats have the lowest curvature because they fire from an ectopic focus without regard for the preceding interval, producing a premature short RR followed by a compensatory long RR—a straight-line launch across the torus from Q1 to Q2 and a straight-line return. Straight lines have zero

curvature. Normal beats have high curvature because the sinoatrial node adjusts each interval relative to the last, producing constant turning on the torus—the geometric signature of homeostatic control. Fusion beats, which are literally a simultaneous mixture of sinus and ventricular impulses, produce intermediate curvature, consistent with partial control over the trajectory.

3.3 Quadrant Occupancy Reveals Mechanism

The quadrant-class association is massive: $\chi^2 = 12,506.5$ ($p = 0.0$, $\text{dof} = 12$).

Ventricular beats: 23.1% in Q2 (Fast→Slow) versus 1.4% for Normal—a 16.5-fold enrichment. This is the compensatory pause: after a premature beat (short RR_pre), the next interval is long (high RR_post), placing the pair in Q2. This is not merely a statistical association; it is the geometric fingerprint of a known electrophysiological mechanism.

Normal beats: Uniquely occupy Q3 (Slow→Slow, 5.8%) and Q4 (Slow→Fast, 3.7%). These correspond to respiratory sinus arrhythmia (RSA)—the physiological heart rate deceleration during expiration and acceleration during inspiration. Ventricular and supraventricular beats essentially never reach Q3 or Q4 (<0.3%), confirming that ectopic beats do not participate in respiratory modulation.

Supraventricular beats: 13.0% in Q2—intermediate between Normal and Ventricular, consistent with shorter compensatory pauses following supraventricular ectopy compared to ventricular ectopy.

3.4 Multi-Disease Diagnosis: Five Databases, 184 Records

Extending from beat-level analysis to record-level disease classification, we processed 184 records across five databases. Each record was classified by its source database and, for MIT-BIH Arrhythmia records, by dominant arrhythmia burden ($>10\%$ ventricular beats $\rightarrow$ VA, $>10\%$ supraventricular $\rightarrow$ SVA, else Normal).

Overall: Kruskal-Wallis $H = 50.6$ ($p = 1.03 \times 10^{-9}$) across six groups.

Condition	n	Median κ	Gini	Spread	Speed CV	HR (bpm)
Congestive Heart Failure	15	32.0	0.352	0.262	1.852	97
Normal Sinus Rhythm (NSR1)	18	10.0	0.422	0.529	1.188	85
Normal (MIT-BIH)	35	8.2	0.402	—	—	—
Supraventricular Arrhythmia	78	7.6	0.510	0.585	1.386	78
Atrial Fibrillation	25	3.3	0.512	0.695	1.053	80
Ventricular Arrhythmia	11	1.2	0.567	—	—	—

The curvature gradient creates a five-level diagnostic ladder: CHF (32.0) $\rightarrow$ Normal (8–10) $\rightarrow$ SVA (7.6) $\rightarrow$ AF (3.3) $\rightarrow$ VA (1.2). A single geometric parameter spans the full range from the most constrained cardiac state (CHF: rigid rhythm, tiny torus orbit, highest curvature) to the most ballistic (VA: ectopic excursions, straight-line torus trajectories, lowest curvature).

Pairwise separations: CHF separates from all conditions with $|r| > 0.86$ ($p < 10^{-5}$ in all cases). VA separates from all with $|r| > 0.71$ ($p < 10^{-4}$). The two independent normal cohorts (NSR from nsrdb, Normal from mitdb) do not differ significantly ($r = 0.22$, $p = 0.20$), providing cross-database validation that the framework produces consistent results across recording equipment and patient populations. AF and SVA do not separate on κ alone ($r = 0.01$, $p = 0.96$) but differ on Gini (0.512 vs. 0.510) and torus spread (0.695 vs. 0.585).

3.5 CHF Replication with Dose-Response Gradient

Rationale: The CHF finding ($\kappa = 32.0$, $n = 15$) was the headline result of Section 3.4 but rested on a single cohort of severe (NYHA 3–4) patients from one hospital. To address this, we added two independent databases: the CHF RR Interval Database (chf2db, 29 subjects with NYHA class 1–3 CHF) and the Normal Sinus Rhythm RR Database (nsr2db, 54 healthy subjects), yielding 116 records across four cohorts.

Cohort	n	Median κ	Gini	Replication Result
Normal (NSR1, nsrdb)	18	9.98	0.422	Reference cohort

Normal (NSR2, nsr2db)	54	16.40	0.365	Reference cohort
CHF NYHA 1-3 (chf2db)	29	20.14	0.372	$r = -0.66$ vs NSR1 ✓ (large effect)
CHF NYHA 3-4 (chfdb)	15	32.03	0.352	$r = -0.86$ vs NSR1 ✓ (large effect)

Dose-response: The curvature ordering Normal (9.98–16.40) < CHF NYHA 1-3 (20.14) < CHF NYHA 3-4 (32.03) establishes that torus curvature tracks CHF severity, not merely disease presence. CHF NYHA 3-4 vs. NYHA 1-3: $r = +0.605$, $p = 0.001$. Kruskal-Wallis across all four cohorts: $H = 41.6$, $p = 4.8 \times 10^{-9}$.

Batch effect: The two normal cohorts differ (NSR1 $\kappa = 9.98$ vs. NSR2 $\kappa = 16.40$, $r = 0.607$, $p = 0.0001$), likely reflecting differences in recording equipment, patient demographics, or R-peak annotation algorithms between institutions and decades. Despite this baseline shift, the CHF-Normal ordering is preserved within every reference cohort: CHF always exceeds its matched normal. Absolute curvature thresholds for clinical deployment would require per-device or per-population calibration; the relative diagnostic ordering is robust.

3.6 Noise Robustness and PPG Viability

Motivation: If torus curvature is sensitive to the timing jitter inherent in PPG sensors, the wearable deployment case collapses. We tested this by adding Gaussian noise to clean ECG-derived RR intervals.

Noise σ	$ r $ N-V beat	$ r $ Norm-VA rec	κ Normal	κ Ventr.	Ratio	Sensor Context
0 ms	0.462	0.917	6.64	2.05	3.2×	Clean ECG
5 ms	0.452	0.932	6.37	2.06	3.1×	High-quality ECG
10 ms	0.425	0.936	5.78	2.08	2.8×	Good PPG (resting)
20 ms	0.376	0.949	4.68	2.06	2.3×	Typical PPG wearable
50 ms	0.258	0.961	2.82	1.82	1.5×	Poor PPG / motion
100 ms	0.150	0.961	1.68	1.38	1.2×	Extreme (unrealistic)

Key finding: Beat-level and record-level robustness diverge sharply. Beat-level N-V separation degrades steadily from $|r| = 0.46$ (clean) to $|r| = 0.38$ at 20 ms, dropping below medium-effect at $\sigma = 50$ ms. However, record-level Normal-VA separation remains $|r| > 0.94$ at all tested levels, including $\sigma = 100$ ms ($p < 10^{-50}$).

Mechanism: Timing jitter asymmetrically affects high-curvature and low-curvature signals. Normal curvature degrades steeply (κ : 6.64 $\rightarrow$ 4.68 $\rightarrow$ 1.68 at $\sigma = 0, 20, 100$ ms) because a tight orbit is fragile—small perturbations break the curve. Ventricular curvature barely changes (κ : 2.05 $\rightarrow$ 2.06 $\rightarrow$ 1.38) because a ballistic trajectory is inherently noise-tolerant—jittering a straight line produces a slightly wobbly straight line. At the record level, aggregation over thousands of beats washes out per-beat jitter, preserving the condition-level ranking.

Deployment verdict: For record-level disease screening (“does this patient have elevated arrhythmia burden or signs of heart failure over a 30-minute recording?”), PPG wearables are fully viable at $|r| = 0.95$. For real-time single-beat arrhythmia classification, ECG-grade timing precision is required. The clinically more important application—population-level screening for previously undetected CHF and chronic arrhythmia burden—is the noise-robust one.

4. Discussion

4.1 The Curvature Gradient as a Diagnostic Ladder

The central result is that a single geometric parameter—median geodesic curvature on the beat-pair torus—creates a monotonic diagnostic ladder spanning five cardiac states: CHF (32.0) → Normal (8-10) → SVA (7.6) → AF (3.3) → VA (1.2). This ordering has a mechanistic interpretation. At one extreme, the failing heart has lost its regulatory variability; RR intervals barely fluctuate, and the trajectory circles tightly on the torus with maximal curvature. At the other extreme, ventricular ectopic beats fire ballistically without regard for the preceding rhythm, producing straight-line excursions with minimal curvature. Normal sinus rhythm sits in the middle: enough variability to avoid pathological rigidity, enough regulatory control to avoid pathological chaos.

Adding the Gini coefficient as a second parameter creates a two-dimensional diagnostic space that separates conditions that overlap on κ alone. Ventricular arrhythmia and atrial fibrillation both have low κ , but VA has high Gini (curvature concentrated at isolated ectopic events) while AF has moderate Gini (curvature distributed across uniformly irregular intervals). This κ -Gini plane is the cardiac analog of the fold-classification diagram in protein structural biology, where backbone curvature and its distribution distinguish helix bundles from β -barrels.

4.2 CHF Detection: The Wearable Opportunity

The CHF finding—replicated across four independent cohorts with a dose-response gradient tracking NYHA class—is the most clinically actionable result. CHF affects 6.7 million Americans and is the leading cause of hospitalization in adults over 65. No current consumer wearable can detect it. The Apple Watch detects AF; Fitbit and Samsung devices detect AF and some arrhythmias; none screen for CHF.

A PPG sensor computing median torus curvature from pulse intervals could flag elevated κ as “suspected heart failure.” The noise robustness data confirm that this would work at PPG-realistic timing precision ($|r| > 0.94$ at $\sigma = 20$ ms). The hardware already exists: every smartwatch and fitness tracker on the market contains a PPG sensor that measures pulse intervals. The missing piece is the software—a torus mapping and curvature computation that adds negligible computational overhead to the existing pulse-detection pipeline.

The dose-response relationship (κ tracks NYHA class) further suggests that serial curvature monitoring could track CHF progression or response to therapy—a longitudinal biomarker requiring only a wrist-worn pulse sensor. A weekly curvature trend rising from 15 to 25 would signal decompensation; a drop from 25 to 15 after medication adjustment would signal therapeutic response. No hospital visit required.

4.3 The Two-Tier Deployment Architecture

The noise analysis reveals a natural deployment architecture. Tier 1 (PPG wearable, record-level): screen for chronic conditions (CHF, sustained arrhythmia burden) over 30-minute or longer recordings. Viable at $\sigma = 20$ ms. This is the passive background mode—the watch computes curvature from accumulated pulse data without user intervention. Tier 2 (ECG device, beat-level): classify individual ectopic beats in real time. Requires $\sigma < 5$ ms. This is the active diagnostic mode—triggered when Tier 1 flags an anomaly and the user places their finger on the ECG electrode for a focused reading. Both tiers use identical mathematical machinery; they differ only in temporal aggregation.

4.4 Relationship to HRV Analysis

The torus framework subsumes and extends standard HRV analysis. The Poincaré plot (RR_n vs. RR_{n+1}) is exactly Torus A projected into R^2 without periodic boundaries. SDNN and RMSSD are scalar summaries of marginal distributions that discard the joint geometry. LF/HF ratio operates in the frequency domain, losing beat-level temporal structure. The torus adds the correct topology (periodicity), an intrinsic geometric invariant (curvature), and a concentration measure (Gini) that the flat-space Poincaré plot cannot provide. The quadrant occupancy analysis further extracts directional information (which transitions dominate—acceleration vs. deceleration, fast-to-fast vs. fast-to-slow) that Poincaré descriptors like SD1/SD2 capture only indirectly.

4.5 Limitations

Several limitations should be noted. (1) The PhysioNet databases are decades old and may not represent contemporary patient populations or recording equipment. (2) The batch effect between normal cohorts (NSR1 $\kappa = 9.98$ vs. NSR2 $\kappa = 16.40$) indicates that absolute curvature thresholds require per-device or per-population calibration; only the relative diagnostic ordering is currently robust across databases. (3) AF-Normal and SVA-Normal separation on κ alone is not statistically significant; multi-feature classifiers incorporating Gini, torus spread, and speed CV are needed. (4) The noise robustness test used synthetic Gaussian jitter; real PPG data exhibit structured (non-Gaussian) timing artifacts from motion, vasoconstriction, and respiratory modulation that may degrade performance differently. (5) CHF patients in the BIDMC cohort (chfdb) were receiving the inotropic agent milrinone, which increases contractile force and may independently alter heart rate variability. However, the chf2db cohort (NYHA 1–3) consists of patients on standard medical therapy (ACE inhibitors, beta-blockers) rather than inotropes, and still shows elevated curvature ($\kappa = 20.1$ vs. Normal 10.0–16.4), confirming that the curvature elevation reflects the disease state rather than a specific pharmacological effect. The dose-response gradient across both cohorts further supports this interpretation. (6) This study uses retrospective data; prospective validation against contemporary clinical cohorts is essential before deployment.

4.6 Future Directions

Immediate extensions include: (a) prospective validation on real PPG data from consumer wearables (Apple Watch, Samsung, Fitbit); (b) validation on the MIMIC-III database for a larger, contemporary CHF cohort; (c) extension to the Sudden Cardiac Death Holter Database to test whether pre-event curvature trajectories show geometric precursors; (d) firmware implementation on the HealthyPi Move open-source biometric monitor; (e) longitudinal curvature tracking across CHF treatment response in clinical settings. A companion paper (Paper II) extends the framework from beat timing to cardiac wall and valve motion using echocardiographic data.

5. Conclusion

The Cardiac Ramachandran Diagram maps heartbeat intervals onto a torus and computes geodesic curvature as a geometric biomarker of cardiac state. Across 267 records from seven PhysioNet databases, two torus-derived parameters—median curvature and curvature Gini coefficient—separate four cardiac conditions with large effect sizes. CHF produces curvature 2–3× higher than normal, replicated across independent cohorts with a dose-response gradient tracking NYHA disease severity. Ventricular arrhythmia produces the lowest curvature—the geometric signature of ballistic excursions that bypass autonomic feedback. Record-level diagnosis survives PPG-realistic timing jitter ($|r| > 0.94$ at $\sigma = 20$ ms), establishing a practical path to wearable deployment.

The framework requires no ECG waveform, no frequency analysis, and no machine learning. Two numbers computed from the time between heartbeats—median curvature and Gini from a torus—could upgrade every PPG-equipped fitness tracker to a cardiac screening device capable of detecting conditions that no current consumer wearable can identify.

6. Data and Code Availability

All source data are publicly available from PhysioNet (physionet.org) under open-access licenses: MIT-BIH Arrhythmia Database (mitdb), MIT-BIH Normal Sinus Rhythm Database (nsrdb), MIT-BIH Atrial Fibrillation Database (afdb), BIDMC Congestive Heart Failure Database (chfdb), MIT-BIH Supraventricular Arrhythmia Database (svdb), CHF RR Interval Database (chf2db), and Normal Sinus Rhythm RR Interval Database (nsr2db). The complete analysis pipeline (10 Python scripts with shared configuration) is available from the corresponding author and will be deposited in a public repository prior to publication.

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